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POLIOMYELITIS OF THE ADULT.

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The occurrence of a true poliomyelitis in the adult, with acute onset under conditions suggesting an infection, is no longer disputed. Such cases have been reported by Gombault[1], Bernhardt[2], Cornil and Lépine[3], Williamson[4], Grawitz[5], Niedner[6], Jagiç[7], Hoppe[8], Caverly[9], and others. The confusion regarding the relationship of this affection to others, notably acute ascending paralysis (Landry) appears to be increasing rather than diminishing. The following report is made because relatively few cases with autopsy have been published, and also in the hope that the matter of classification in view of our growing knowledge of the pathological anatomy is of sufficient interest to permit a reopening of the general subject.

Case I.—J. C. L.,* an American by birth, unmarried, twenty-five years old, worked chiefly indoors, lifting. He had been especially overtaxed just before his present illness, but had not been exposed to cold or similar influences. His family history was good, and his personal history showed no undue excesses in tobacco, alcohol or stimulants of any sort. He had had gonorrhea six years before this illness, followed three or four years later by a slight stricture. He had also had soft chancres, but sufficient evidence of syphilis was lacking. He had not suffered from rheumatism. His present illness began September 11, 1898. The onset was, to him, similar to previous experiences in taking cold. There was headache, backache, pain in the extremities, without sense of fever or chilliness. During that day he grew worse and he had a night of poor sleep. On the following day (Monday), he felt no better and could eat little. Thinking he had "grippe" he started to "walk it off" and kept on his feet the greater part of the day. His legs felt as well as they ever had, but his back was weak and gave him considerable pain when he suffered a jar, as in stepping off a curbing. He ate no dinner, but took a drink of whiskey, went to bed early and slept well. The next morning (Tuesday), he rose early to get whiskey, but immediately fell; he pulled himself to his feet by the aid of the bedpost, but found that he could not stand. He was, however, able to creep. He got his drink and went back to bed. He then found that he could not move his feet or toes. He had some nausea and vomited. A physician was called, who found the temperature to be 103.5°. He was forthwith sent to the Boston City Hospital in the service of Dr. E. M. Buckingham.

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*For the following clinical history I am indebted to Dr. E. M. Buckingham, Boston City Hospital, Dr. J. J. Minot, Carney Hospital, and Dr. Pillsbury, formerly a student of medicine at the Harvard Medical School.

The following day (Wednesday), both thighs were paralyzed; he could not sit up nor turn in bed. The next day his arms were weak, particularly the right, and he was unable to breathe deeply, except by grasping the head of the bed and thereby bringing the accessory muscles into play. Sensation, he thought, was at no time lost in any part of his body, but there was at first a slight feeling of numbness in the limbs; they felt alternately warm and cold, and were often covered with sweat. This was especially noticeable on the right leg. The paralysis was flaccid from the first.

For the first seven days after entrance, the bowels moved only by enema; the catheter was used for two days; after that there was no difficulty with bowels or bladder. The temperature remained elevated for four days, became normal two days later, and remained so. The special senses were at no time disturbed; the appetite in general was good, and sleep was satisfactory. For about a week after entrance, there was some pain in arms and legs. His back ached when moved, otherwise not. The muscles of face, neck, deglutition and mastication were not affected.

About a week after entrance to the hospital and about ten days after the onset of the disease, voluntary movements of the arms became stronger, and the breathing less labored. A week later he began to regain slight control of his toes and feet and soon after of his hips. After November 1 he began to sit up an hour a day, supporting himself in an erect position by aid of his arms. His back remained very weak.

The physical examination on entrance showed no Widal reaction and no evidence of plasmodia in the blood. The urine had a specific gravity of 1023, without albumin or sugar, or anything significant in the sediment. November 9, pupils and ocular muscles were normal. The teeth were in good condition and showed no evidence of metallic poisoning. Cranial nerves were in general free of involvement. The chest was thin, movements feeble, but equal on the two sides; the accessory muscles were not used. Inspiration was feeble and prolonged; respiration was 28 and regular. Palpation, auscultation and percussion showed nothing abnormal. The heart was normal with a somewhat rapid pulse. The abdominal organs were negative. The arms were thin and the muscles flabby, the skin warm, moist and of natural appearance. Voluntary movements, both of flexion and extension, could be weakly made. The left hand-grasp was particularly weak. The legs were thinner and the muscles more flabby than in the arms; the skin was normal. Voluntary movements at the hips very weak; the flexors of the legs could be seen to contract, but not with sufficient strength to induce the slightest flexion of the knees. Motion of the feet and toes was possible, but weak. Passive motion was not limited in any joint. The knee-jerks were absent.

Sensation for contact, pain and temperature was not impaired. There was tenderness on either side of the vertebral column in the region opposite the angles of the scapulæ, and in the axillæ and popliteal spaces, without marked muscular tenderness. No notes of an electrical examination are obtainable.

November 11, the patient contracted a cold, during which he had difficulty in expectoration and respiration; his cough was feeble; the breathing was vesicular, with numerous râles. No tubercle bacilli were found in the sputum. November 14, the cough had diminished, respiration was easier and the lung condition decidedly improved. Slight improvement in movements also occurred and a very slight knee-jerk was obtained. There was no sign of contracture, beyond a tendency to keep the knees flexed.

By his own request the patient was discharged from the

City Hospital, and admitted to the Carney Hospital in the service of Dr. J. J. Minot.

November 22, examination showed essentially what has already been described. The knee-jerk was reported as again lost, with very slight plantar and cremasteric reflexes. Atrophy of legs and thighs was noted, and very limited movements. A fundus examination showed nothing abnormal. The patient was hopeful, and thought the movements of the legs were slowly improving. A certain amount of flexion at the right knee was possible, as well as movements of the feet and toes.

December 7 there was complaint of dyspnea which increased, though apparently not to an alarming degree. At 4 A.M., December 8, he appeared to be comfortable; when next seen an hour and a half later he was dead. His temperature had remained normal during his stay in the hospital.

This case presents a typical picture of poliomyelitis as usually met with in the adult; unexplained ill feelings with rise of temperature, followed by paralysis, involving all four extremities, but predominantly the legs, which reaches its height in one to three days, and then tends towards improvement; some subjective disturbance of sensation, passing in a few days, leaving a condition essentially of motor paralysis of the muscles of the trunk and extremities, without at any time disturbance of consciousness.

Autopsy—By Dr. G. B. Magrath, Dec. 8, 1898; six to seven hours after death. Other organs than the central nervous system showed nothing of importance. The brain, cord and a portion of the right sciatic nerve were preserved for microscopic examination. Portions of the brain—paracentral lobules—and of the oblongata and cord were hardened in alcohol for later Nissl staining. The remainder of the cord was preserved in 10 per cent. formalin, and Müller's fluid, the sciatic nerve, in Müller's fluid, and the brain in 5-10 per cent. formalin. The brain was normal in external appearance, except for slight adhesions between the dura and pia at the tip of both temporal lobes, but much more marked on the left. The cord also was normal externally, but on section at various levels, there was a very striking injection of the ventral horns, throughout the cord, marking them out sharply from the surrounding white matter. A culture made from the subdural space of the cervical region gave no bacterial growth.

Microscopic Examination—A study of the cord at various levels shows throughout its entire extent an almost complete distintegration of the ventral gray matter, extending to a considerable extent into the dorsal horns, and slightly into the surrounding white matter, confirming the appearances seen macroscopically. A more detailed study of the cord from below upward gives the following appearances: A section through the conus terminalis shows an exceedingly slight accumulation of cells in the neighborhood of blood vessels; otherwise there is at this level no abnormality. In the sacral cord and lower portion of the lumbar enlargement the evidences of inflammation become more apparent. The perivascular spaces are in many areas infiltrated with extravasated cells, both in the ventral and dorsal horns. A Weigert section shows a clearly marked, though not complete loss of myelinated fibers in the ventral horns, loss of many nerve cells and partial degen-

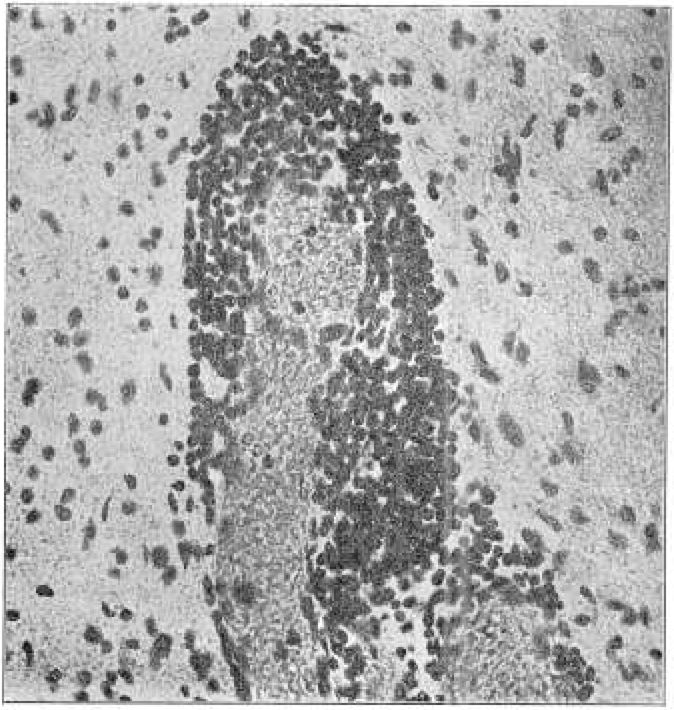

Fig. 1. Infiltration of a perivascular space with extravasated cells.

eration of ventral roots. The blood vessels throughout the section, notably in the pia and nerve roots are completely distended with blood, but nowhere except in the ventral horns and portions of the dorsal horns does there appear to have been an exudate from the vessels. The cross-section of dorsal nerve roots at this level shows in several places small areas of necrosis which have the appearance of dependence upon local vascular disturbances.

At a slightly higher level in the lumbar enlargement the pathological process is very much more extensive, and the appearances here will serve as a type of the character of the alterations throughout the cord. A hematoxylin-eosin specimen shows the end results of a violent inflammation. The same conditions, in general, prevail as those already described, but much greater in degree. The ventral horns are filled with distended capillaries in the neighborhood of which are many extravasated cells. It is noticeable that among the cells of this exudate no leucocytes are to be seen, which is not remarkable in consideration of the fact that three months had elapsed from the acute onset

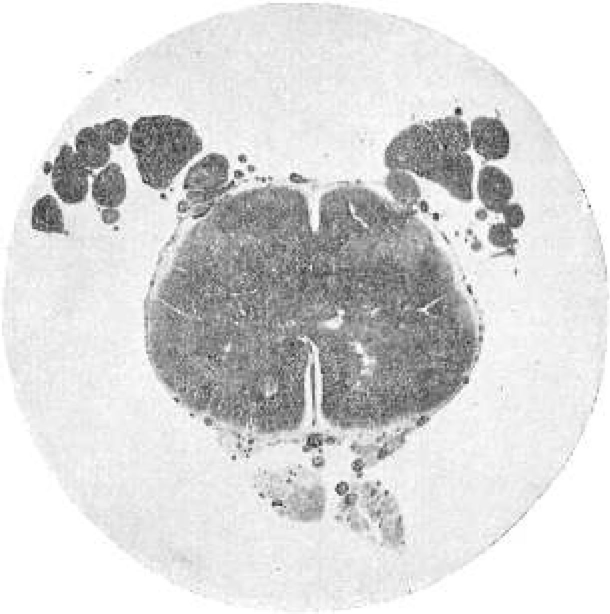

Fig. 2. Section stained by the Weigert method, showing partial degeneration of ventral roots.

of the disease to the death of the patient. The nuclei of the remaining cells are for the most part small, round, deeply staining with hematoxylin; in general of the lymphoid variety. Occasional, elongated, somewhat vesicular nuclei may be seen; it is possible that many, or perhaps all of these nuclei may belong to proliferating neuroglia cells, as part of the reparative process; no mitoses, however, are discoverable. The general dilatation of capillaries extends distinctly into the dorsal horns and to a less degree into the white matter and vessels of the pia. The lesion is, however, so much more marked in the ventral horns, that that may be regarded as its essential location.

Sections stained by the Nissl method are of particular interest. The appearances in the lumbar region may again serve as a type of the changes found throughout the cord. Most noteworthy is the diminution in the number of nerve cells. At one of the levels studied in the lumbar enlargement not a single large ventral horn cell is to be seen on one side, while on the other the number is markedly reduced. The cells destroyed by the original inflammatory process have for the most part entirely disappeared, while the relatively few remaining cells are frequently normal as regards their markings, a condition to be expected in a local traumatic process as poliomyelitis may properly be regarded, so far as the nerve cells are concerned. Occasional cells may be seen, showing marked chromatolysis, and distinct dislocation of the nucleus, having the general appearance of cells undergoing the so-called degeneration at a distance, a type of degeneration which might easily occur in a destructive process of this sort. It is often difficult to trace certain processes to a distance from the cell body, and in some instances the dendrites appear as if broken, probably by the violence of the original exudation. There is no sufficient evidence of cell disintegration due to a slowly acting toxic cause. The point of chief interest in the appearances given by the Nissl method is the comparative lack of transitional forms between a complete destruction of nerve cells and wholly uninjured cells.

There is no distinct evidence of the process described by Marinesco as restitution of injured cells, possibly owing to the insufficient time which had elapsed since the beginning of the illness. Swollen cells with abnormally deeply staining chromatin are not visible, although many cells show an unusually definite coloration of the Nissl bodies.

The changes described for the lumbar region extend throughout the cord with essentially unvarying intensity, and therefore require no special comment. In the cervical region a few large cells remain intact and a considerable number of smaller cells, faintly staining and much degenerated, may be seen on careful search with a high power. The vascular changes are as pronounced here as at other portions of the cord.

Slightly higher, in the lower oblongata, the vascular distention and exudation have almost wholly disappeared. The mid-oblongata at the level of the hypoglossus nucleus shows no abnormality. The cells are well stained and sufficient in number. The upper oblongata, pons, cerebellum and cerebral cortex are normal, beyond an occasional extreme distension of blood vessels, to which no special pathological significance is to be attached. There is nowhere evidence of exudation from the vessels. Beyond the distension of the vessels, especially marked in the cord, the walls show no abnormality. The sciatic nerve is very markedly degenerated. Certain of the bundles have almost completely lost their myeline (Weigert); whereas others remain essentially intact. This observation is of some value as showing the relative arrangement of motor and sensory nerves in the peripheral trunks. It is also of interest that sections stained by Marchi's method show no degeneration of the myeline, probably due to the fact that three months had elapsed since the onset of the disease.

In summarizing the chief alterations observed the following points are to be noted: Essential limitation of the pathological process to the spinal cord; in the cord, chief though not exclusive localization in the ventral horns; evidence of primary inflammation with secondary degeneration and destruction of nerve cells; marked degeneration of peripheral nerves.

The clinical course and pathological anatomy of this case correspond closely to several others which have been reported within the last few years, and to which allusion will later be made.

Case II.—A girl of seventeen, up to that time well and athletic, was taken ill with vague symptoms, referable to her head with some pain about the body; she complained of dizziness and general weakness. This condition persisted until she was forced to take to her bed at the end of about three weeks, with an acute febrile attack, the temperature at one time reaching 103.8. During this acute attack, or immediately following it, a rapid loss of power in the arms and later in the legs developed, which became practically a total paralysis, accompanied by a somewhat sighing respiration.

The patient vomited easily and often; there was retention of urine and feces, necessitating the use of the catheter and enemata. There was also temporarily excessive pain in the legs, of a lancinating character.

Physical examination made soon after the onset of the paralysis showed complete motor disability in both legs and in the right arm. The left arm was also practically helpless, except for some flexion at the elbow and wrist; extension was impossible. The cranial nerves were not involved, nor was the phrenic to a noticeable degree. Sensation was uninvolved, except for a possible slight hyperesthesia of the legs; there were no pains as at first. The deep reflexes were lost; the temperature had returned to normal and the general condition was much improved over what it had been. The urinary and rectal disturbance, however, persisted.

At a second examination, made about a month later, this loss of control of the sphincters had passed; it had not been necessary to use the catheter after the first week of the onset of paralysis. There was in other respects also, some improvement; more motion of the left arm was possible, but it was still very weak. The right arm was slightly movable at the shoulder joint only. The right leg remained absolutely paralytic; likewise the left leg, except for slight movement of the toes. The muscles were flabby and showed evidence of atrophy. Sensation remained essentially unimpaired, and the sharp pain of the earlier stage had entirely disappeared. Respiration was now unaffected, and the mental state was clear.

The patient later, fortunately, came under the treatment of Drs. E. H. Bradford and E. G. Brackett, of Boston, who through assiduous training of the muscles, over a long period of time, were able to bring about a degree of improvement which it is not to be supposed that nature unaided could have done. Nearly two years after the onset of the disease, Dr. Bradford writes that the patient is able to stand and take a few steps with the help of crutches, and that progress had been made during the last six months.

The following case, occurring in a boy shortly before puberty, is of importance as illustrating a cerebral complication or accompaniment of the disease which is not altogether easy of explanation, except through association with an accompanying encephalitis.

Case III—N. A., a boy, fourteen years old, came to the Nerve O. P. D. Massachusetts General Hospital (No. 22,174), March 31, 1902, with the following history: Toward the end of August, 1900, after a bicycle ride of about fourteen miles, not associated with special fatigue, and after wading a few hours later, he felt chilly; he was not ill that night, but the next day had severe backache and headache. The following day he had considerable pain in the right foot. On waking the next morning, after a good night's sleep, his right leg was paralyzed. That afternoon the left leg began to grow weak and the next morning it also was paralyzed. On this day fever was first noticed, though it had probably existed for several days, and he began to grow somnolent. He rapidly sank into a stuporous condition from which he could not be roused by any ordinary stimuli, and remained so for about eight weeks. He did not speak during this time, except on one occasion, about two weeks after the onset, when he is said to have conversed intelligently for about half an hour. After that he became more stuporous with increased fever. His bowels and bladder acted involuntarily, but he was always able to swallow. There was at no time paralysis of any of the cranial nerves; the father gives an extremely doubtful history of retraction of the head. Recovery from this condition was gradual, with increasingly prolonged intervals of consciousness. The patient remembers absolutely nothing of this period of eight weeks; his mental state is now entirely normal. He had had no suppuration of the ears; and beyond chicken-pox the previous spring and headaches with constipation, he had previously been well. An analysis of the drinking water showed the prescence of lead, and a slight amount was also found in the urine.

After recovering from the stuporous condition spoken of above, all four extremities were more or less paralyzed, the legs to such an extent that he has not since been able to stand or walk, and the arms to a less degree. There has been no disorder of the sphincters nor of sensation.

Physical examination at the present time shows the following conditions in detail: General appearance of health: the patient is well grown and of good color. The head and cranial nerves show no abnormality whatever. The pupils are large, equal and respond to light. The fundus shows nothing pathological. The patient has a rachitic chest. All the muscles are soft and many show distinct atrophy. The trapezii and sternomastoids are not involved. Movements of the shoulders are somewhat weak; the muscles of the upper arms are flabby, but individual motions are preserved. The right arm shows greater atrophy than the left. In both arms there is distinct atrophy of forearm muscles on the ulnar flexor side. The hands are similarly atrophic, but more marked on the right. The small muscles of the thumb are particularly involved, permitting but very slight movement on the right. The interossei are somewhat affected. Extension of hands at the wrist is possible. In respiration the movements of the ribs are imperfect. There is some lordosis. Right glutei completely paralyzed; left approximately normal. The legs are much more atrophic than the arms, practically all the muscles taking part. The patient as stated, cannot stand, but creeps with comparative ease, flexing the legs both at hips and knees. In the sitting posture he cannot raise either leg at the hip nor extend the leg on the thigh. If knees are supported, flexion and extension are slightly possible. Abduction and adduction at the thighs imperfect, particularly on the right. All movements of the right foot are abolished; extension of toes of the left foot possible; flexion lost. Slight flexion and extension of the ankle possible. The feet are cold; the right foot somewhat cyanotic.

Reflexes, superficial and deep, are lost; the paralysis is flaccid; there are no contractures.

Electrical examination shows quantitative diminution to both faradism and galvanism, without qualitative change in the muscles examined.

During the past four years and three months at the Out-Patient Neurological Department of the Massachusetts General Hospital, anterior poliomyelitis has been diagnosticated seventy-six times. Of this number ten occurred in persons upward of twelve years of age; five of these eighteen or more years old. In several of these cases, however, the diagnosis permits of doubt, so that, judging from these figures, the percentage of adult cases, uncomplicated by cerebral symptoms, is very small.

Case IV—L. F., twenty-two, unmarried, a school teacher, at nineteen was taken with an illness supposed to be influenza. On the third day of this illness a sudden paralysis of the legs came on which had prohibited walking since. There was no sensory nor sphincter disturbance; there remained practically a complete paralysis of the right leg, and a very marked paresis of the left. There was double toe drop; the skin of the feet was cold and brawny in appearance. Probably much muscular atrophy, but masked by fat. The knee-jerks were absent.

A typical case of poliencephalitis associated with a cervical poliomyelitis occurred during this period in which the cerebral symptoms so far predominated that it is rather to be considered in another category than poliomyelitis.*

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*This case will be alluded to later in this paper and reported in detail in another place. See in this connection, Sherman and Spiller: "A Case of Poliencephalomyelitis in an Adult." Phil. Med. Jour. v. 734, 1900.

Symptomatology.—The cases described above conform to the generally accepted picture of acute poliomyelitis. Had they occurred in infants or young children, no doubt whatever would have been entertained of the diagnosis. A febrile attack, with general constitutional symptoms, followed by a more or less extensive paralysis of one or more extremities of a purely motor type, rapid in onset, forms a characteristic picture at whatever age it occurs. In spite of an increasing number of clinical and some pathological observations showing conclusively the existence of acute poliomyelitis in the adult there still remains a certain prejudice against the acceptance of the diagnosis, so deeply rooted is the feeling that it is peculiarly a disease of childhood. It is unquestionably a rare affection after the fifteenth year, but no one who has studied the reported cases can for a moment deny that it occurs in characteristic form, and probably with much greater frequency than ordinarily supposed. Bernhardt[11] in 1874 reported three cases in men, two of which involved all four extremities. He notes the frequency of this extensive involvement in the adult, an observation made and confirmed by others since. In 1875, Cornil and Lépine[11] reported with autopsy a case of relatively slow onset, with characteristic cord changes, which others might have been inclined to regard as a case of so-called Landry's paralysis. Williamson's[12] case was a man of twenty-two, who died five weeks after a sudden paralysis of both arms and legs, without permanent sensory disorder, and in which the autopsy confirmed the diagnosis. Grawitz[13] narrates the case of a woman of eighteen, with involvement of all four extremities, followed by partial recovery, the paralysis in this case being of somewhat slow onset, occupying several days to reach its maximum. A somewhat similar case in a man of nineteen, followed by death with paralysis of respiration is reported by Niedner[14], without autopsy. Jagiç[15] observed a case of primary involvement of the leg, extending to one arm, followed by death in which the similarity to Landry's symptom-complex is noted. Hoppe's[16] cases, although not fatal in outcome, admit of no reasonable doubt in diagnosis, and Caverly's[17] recurring in the famous Rutland, Vermont, epidemic of 1894, are equally beyond suspicion, although no autopsies were obtained. Of special interest on the clinical side are those cases which somewhat arbitrarily have been classed as Landry's paralysis, at times in spite of lesions found post-mortem which would naturally place them in the category of anterior poliomyelitis. Among these are the observations of Bailey and Ewing[18], J. J. Thomas[19] and Greene, Wilson and Rothrock[20], all of whom describe lesions (see remarks on pathological anatomy) essentially characteristic of poliomyelitis, and yet classify their cases under the vague heading of Landry's paralysis. The tendency to consider cases of rapidly advancing paralysis, whether fatal or not, and regardless of the pathological findings as Landry's paralysis is very noticeable in much of the best recent work in America. An attempt to fit pathological changes into a preconceived clinical picture usually leads to an unnecessary confusion, and such has been the result here. We have elsewhere[21] discussed what to us seems the proper attitude to assume toward the complex of symptoms which goes by the name of Landry. We wish, now, merely to reiterate that the clinical course of the disease must not be the sole criterion of its nature. Poliomyelitis is none the less poliomyelitis because it simulates in its clinical course what Landry many years ago described as an acute ascending spinal paralysis. German observers have on the other hand been quick to recognize the occurrence of a form of poliomyelitis in the adult, in which the paralysis has been of relatively gradual onset and often ascending in type. In 1876 Schultze[22] reported a case of poliomyelitis with autopsy in which there was a resemblance to a rapid Landry's paralysis. Again in 1883, Schultze[23] writes of two cases under the title of "Ascending Atrophic Paralysis with Fatal Outcome," one in a man of 44. and the other in a man of 30. Immermann[24] in 1885 reported an important case with autopsy. The patient, a man of 22, had an ascending flaccid paralysis of the legs, followed by involvement of an arm and later bulbar symptoms. The autopsy showed changes in the ventral horns. Immermann assumes from this case that a form of anterior poliomyelitis exists which corresponds to the clinical picture of Landry's paralysis.* Various

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*"Die Landry'sche Paralyse und die acuta Poliomyelitis anterior nur die graduell verschiedenen Bilder *eines* Krankheitsprocesses darstellen." *Loc. cit.*, p. 305.

other writers on the subject note the fact that in many cases the onset of the paralysis is by no means so stormy as in infants, but they are not thereby induced to exclude them from the general category of poliomyelitis, influenced no doubt by the autopsy findings.

Discussion has also arisen with regard to the differential diagnosis between poliomyelitis of the adult and multiple neuritis. In 1880, Leyden[25], in an exhaustive paper, attempted to elucidate the question by a somewhat elaborate classification of the forms in which poliomyelitis may occur. One form he regarded as essentially due to a peripheral neuritis. Very recently Strümpell and Barthelmes[26] have again reverted to the matter in much clinical detail, calling attention to the fact that the diagnosis of adult poliomyelitis has decreased with the advent of the study of polyneuritis. These writers refer to Müller's[27] monograph on acute atrophic spinal paralysis, in which no case is to be unquestionably regarded as an acute poliomyelitis.

Except in rare instances the differential diagnosis on the clinical side should not present many difficulties provided the term neuritis is used in the sense of an inflammation of the peripheral nerve trunks, and not as another term for neurone degeneration. In the latter case various forms of motor neurone degenerations might easily simulate a poliomyelitis, with or without transient sensory disorder. The following cases are of interest in this connection:

Case V.—W. P., a farmer, nineteen years old, came to the Massachusetts General Hospital in March of this year. About a year and a half before, he had gone to bed feeling well. During the night he had considerable pain in the back, which kept him awake. The following day he had headache, for which he remained in bed. In the afternoon, on attempting to walk, he found that his right leg "gave way" at the knee. For the next two or three days both legs were slightly sensitive to pressure, which persisted in modified form for some weeks. Treatment by electricity and massage did no good, and the right leg gradually decreased in size and became sensitive to cold. Examination a year and half after the onset showed a marked paresis of the right leg. The patient could lift it slightly while sitting, but was wholly unable to extend the leg on the thigh. The sphincters were unaffected. There was no venereal or alcoholic history; the right knee-jerk was absent; there was no objective disturbance of sensation.

Case VI.—A man of twenty-nine, a temporary patient at the Massachusetts General Hospital, said that ten years before he had had "rheumatism in his knees," extending to his hips, by which he was crippled for six months. The right leg apparently recovered, but the left has remained weak. There was wasting below the knees, and a complete paralysis of the left foot and toes for a year. The general health was good, and at the time of examination there were no sensory disturbances.

Whatever doubt there may be as to the proper interpretation of these cases, in both of which the diagnosis of poliomyelitis was made, it will be generally admitted that cases I and II are typical of what has ordinarily been called poliomyelitis, and certainly could not be brought into the category of polyneuritis.

Temporary disorders of sensation, often of an extreme degree of severity, have frequently been described. in the early stages of the disease, no doubt to be explained by the involvement of portions of the dorsal horns in the inflammatory process. This should never lead to an error in the clinical diagnosis, particularly after the acute onset has passed.

Pathological Anatomy.—As our study of the pathological anatomy of the nervous system progresses, it is becoming increasingly clear that we must continually widen the bounds of what we have hitherto regarded as individual diseases. This is particularly true of the group of lesions it is now necessary to consider in connection with a process apparently so clearly defined as poliomyelitis. This disease has, perhaps, occupied a somewhat more stable position than many other affections of supposed definite organic basis, but the time has apparently come for a clearer recognition of its relation to various cerebral and peripheral disorders of so dissimilar a character clinically as peripheral neuritis and acute encephalitis or Landry's symptom-complex and acute ophthalmoplegia. Is poliomyelitis occurring either in the child or adult the same disease as these, and due to the same general causes, or are we dealing with totally dissimilar affections both from the pathological and etiological, as well as from the clinical, point of view? This question may, at present, be answered only in part, but much light has been thrown upon it by various recent pathological reports.

Regarding poliomyelitis certain facts are now generally accepted:

1. That it is an infection of undetermined character, limited to the region of the ventral horns of the cord.
2. That the pathological alterations found are consistent with the theory of a primary inflammation and a secondary destruction of nerve-cells.
3. That the ultimate changes may closely resemble or be identical with those observed in primary degenerations of nerve elements.
4. That the symptoms resulting tend to be of sudden onset, capable of some degree of improvement, motor in type and unassociated with permanent sensory disturbance.

Even these fundamental statements have by no means been universally accepted, due, no doubt, in great measure to the widespread influence of Charcot. Following the earlier writers on the subject, Heine, Cornil, Duchenne, Prevost, Cuming and others,* Charcot and Joffroy[29], reporting a case of poliomyelitis twenty-five years after its onset, advanced the theory that the ventral nerve-cells are specifically and primarily involved, differing only from progressive muscular atrophy in being attacked more suddenly. With the material at their disposal this supposition was entirely natural. It is, however, somewhat remarkable that certain writers should later have adhered to this view, in spite of much better opportunities for study. Among these is Rissler[29], who had the exceptional opportunity of studying microscopically five cases, three of which were very recent, occurring in the epidemic described by Medin[30]. For reasons which are not altogether obvious, Rissler concludes that in the recent cases, in spite of cellular exudation in combination with degeneration of nerve-cells, the primary affection is of the nerve-cells. In 1893 von Kahlden[31], unconvinced by many apparently conclusive papers maintaining the existence of a primary inflammation, reverts again to primary degeneration of nerve-cells as the essential lesion. He bases this opinion upon the examination of material from persons in whom the paralysis had been of long standing. In decided opposition to this point of view stands an array of painstaking articles, of which the following are the most important: Roger and Damaschino[32], Schultze[33], Turner[34], Bramwell[35], Drummond[36], Williamson[37], Goldscheider[38], Siemerling[39]. All of these writers and others, reference to whose work will be found in the bibliography, are united in regarding the primary lesion as inflammatory, brought about by an unknown etiological factor, acting in the general distribution of the ventral arteries of the cord. With certain variations in the details of the pathological findings, recent observers are essentially united in this point of view. It is easy to understand that the appearances after the lapse of years might closely simulate those found in processes of quite a different origin; for example, progressive muscular atrophy. The question can only be decided by the examination of relatively recent cases, of which there are now enough at hand to form a final judgment. A matter of much more vital interest, at present, is the determination of the problem whether poliomyelitis represents a disease *sui generis,* or whether it has intimate relationship with others, differing from them only in degree and location, and consequently in symptomatology. A study of these questions is best made in the adult form, since the diseases which poliomyelitis may simulate occur more frequently in adults than in children. Considered from the pathological side, omitting for the present any question of etiology, the relation of poliomyelitis to multiple neuritis, Landry's paralysis, myelitis, encephalitis, poliencephalomyelitis, and progressive muscular atrophy needs to be studied.

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*See Bibliography.

Multiple Neuritis:—On the clinical side the possibility of confusion in the diagnosis between poliomyelitis and polyneuritis has long been recognized and discussed. Allusion has already been made to the important papers of Leyden and Strümpell and Barthelmes bearing on this point. However difficult, at times, the clinical diagnosis may be, the histological changes in the two conditions would hardly admit of question if we regard the lesion of poliomyelitis as in all cases a primary inflammation of the ventral horns within a definite arterial area.

If the term neuritis be applied to peripheral neurone degenerations manifesting themselves only in the axones, no confusion could arise in the histological appearances; if the peripheral nerves show evidence of inflammation without destructive lesions in the ventral horns there is likewise no difficulty. If, on the other hand, inflammatory changes of the ventral horns occur with consequent changes in peripheral nerves, the anatomical diagnosis of poliomyelitis is apparent. So far as we are aware no case has been published in which a coincident inflammation of ventral horns and peripheral nerves has occurred, indicating a common origin. In the absence of such a case it is fair to assume that the etiology of the two conditions is essentially different, a point to which we shall revert later. A mere difference in the degree of strength of the irritant is not sufficient to account for the difference in location of the consequent inflammatory reaction. Redlich[40] on the other hand inclines to the view that poliomyelitis and polyneuritis are the same affection with different localizations.

Landry's Paralysis:—As has been suggested, the tendency to find a pathological anatomy for a group of symptoms resembling in some respects poliomyelitis has led to relatively frequent descriptions of the lesions of poliomyelitis under the heading of Landry's paralysis. Judging from the illustrations, as well as the description of the course of the disease and the lesions, Bailey and Ewing's[41] case is of this character. In this case there was sudden loss of power in both legs, followed the next day by paralysis of one arm and paresis of the other. Sensation was unaffected; the reflexes were lost; there was diminished faradic response in the paralyzed muscles. On the clinical side such a history corresponds perfectly to adult poliomyelitis with cerebral involvement and very imperfectly to the symptom-complex described by Landry. The gradual invasion of muscle groups without electrical changes, supposed to be characteristic of Landry's paralysis did not occur. On the pathological side "the lesion consisted in intense congestion of all the blood-vessels especially of the ramifications of the central branch of the anterior spinal artery," and again, "at some levels congestion and exudation were so intense that nothing could be distinguished but the round-cell infiltration and the detritus of neuroglia and ganglion cells." Various degenerative changes in nerve-cells were also, naturally, found. These are unquestionably the exact lesions of poliomyelitis as described frequently in children by entirely competent observers, and less often in adults. They cannot be brought into the category of Landry's paralysis, unless that vague term be enlarged to include poliomyelitis, which seems manifestly undesirable. Thomas'[42] Case I is likewise a case of acute anterior poliomyelitis in the adult, described in his paper, entitled "Two Cases of Acute Ascending Paralysis with Autopsy." The diagnosis is so made in the description of the case, and a study of the sections, with which Dr. Thomas has kindly provided me, leaves no doubt of the fact that it is not properly classified under Landry's paralysis. A similar case of much interest has been published by Greene, Wilson and Rothrock[43] as "A Case of Landry's Paralysis." There was high temperature, followed by sudden paralysis of legs, quickly extending to arms. Death, after forty-one days of artificial respiration. Microscopic examination showed a violent inflammation in the ventral horns, extending the entire length of the cord, leading the authors to speak of the lesion as a "parenchymatous ascending acute poliomyelitis." In 1875 Webber[44] in a paper on myelitis deprecated the use of the term acute ascending paralysis as misleading. In this connection articles by Schultze[45], Immermann[46], Jagiç[47], Mills and Spiller[48], as well as those quoted above, are of much interest. As expressed in a former paper[49], it seems altogether desirable to abide by pathological findings, unless a better criterion be offered. If this had been done, we should certainly have heard much more of the supposedly rare poliomyelitis of the adult.

Myelitis occurring in its typical form as a more or less complete transverse lesion of the cord of inflammatory character should not often be confused with, or mistaken for, poliomyelitis. That transitional forms of inflammation may occur must be conceded. It has long been recognized that the lesions of poliomyelitis may extend beyond the confines of the ventral horns, but the greatest intensity of the process has usually been fairly limited to those areas (Roger and Damaschino[50], Turner[51], Eisenlohr[52], Rissler[53], Goldscheider[54]. A very important case in this connection is one described at length by J. J. Putnam[55] in 1883. The patient was a woman of twenty-two, who was suddenly attacked with disorders of sensation, followed by muscular wasting, the right arm and hand being especially affected. A diagnosis of adult poliomyelitis was made. Microscopic examination of the cord showed that both ventral and dorsal horns were involved, the lumbar cord being less affected than other regions. The blood vessels were thickened, deeply congested, and there was extravasation of cells. The inflammation was acutest in the white matter, many of the fibers of which showed degenerative changes. The ventral nerve roots were degenerated to a greater degree than the dorsal. There was a moderate amount of leptomeningitis. Putnam concludes from the pathological findings that the disease might better be designated, "diffuse myelitis." It is a matter of regret that the peripheral nerves were not examined. From our present point of view the case could with difficulty be placed in the category of acute poliomyelitis. It illustrates better than any case of which we have knowledge, the extension of an inflammation, very similar in general character to that which occurs in poliomyelitis, over a considerable area of the cord, and goes far toward proving the existence of transitional forms of disease. The coincident occurrence of a meningitis is also important.

Encephalitis and Allied Conditions.—In many cases in which a poliomyelitis is the predominant feature in the clinical picture, cerebral involvements of more or less severity have been described, and many of them confirmed by autopsy. In Case III of this paper, an unmistakable poliomyelitis was associated for about two months at the onset of the disease with a profound disturbance of consciousness, without involvement of cranial nerves which finally disappeared, leaving no defect. The possibility of a mild encephalitis must certainly here be consided, though we have no means of proving its existence. Involvements of cranial nerve nuclei have been reported many times as poliencephalitis, superior or inferior, or ophthalmoplegia, without involvement of the cord. That the character of these various processes is similar is shown by the fact that many carefully studied cases of poliomyelitis, especially in the adult, give evidence of a slighter, but perfectly definite inflammatory reaction throughout a part or whole of the brain stem. Such observations have been made by Gombault[56], Rissler[57], Redlich[58], Greene, Wilson and Rothrock[59], Jagiç[60], Bülow-Hansen and Harbitz[61], and on the clinical side notably by Caverly[62], and Medin[63], who studied the two best recognized epidemics of the disease which have hitherto occurred. In these cases the cord affection was, for the most part, predominant, and what changes were found in the oblongata were regarded as subordinate in intensity. Bailey and Ewing's case of Landry's paralysis, to which we have previously alluded, is of this type; in that case lesions were also found in the cerebellum. A still more definite connecting link between the cerebral and spinal affections is to be found in the rare cases of poliencephalomyelitis, a few of which have been carefully reported with subsequent autopsy. Under the heading "A Case of Poliencephalomyelitis in an Adult, Presenting the Clinical Picture of Landry's Paralysis, and Fatal in Thirty-eight Hours After First Definite Motor Symptoms," Sherman and Spiller[64] have recently reported a characteristic instance of combined cord and brain lesions. The patient, a man of twenty-one, was taken with headache, vomiting and slight rise of temperature. Two days later walking became difficult through weakness of the legs; the next day respiratory paralysis set in followed by weakness of the arms, the legs being almost completely paralyzed. The mind remained clear and sensation was unaffected. Death from paralysis of respiration. The pathological examination showed organisms of the colon group from liver and kidneys; engorgement of vessels of the cord in all regions; cellular infiltration of gray matter, meninges and spinal roots; less in white matter and less in dorsal than ventral horns; hemorrhages in spinal gray matter; degeneration and loss of ventral nerve cells; ventral spinal roots normal. The oblongata was similarly affected, but to less degree; degenerattive changes in nucleus ambiguus; vessels of pons, peduncles and cortex distended, but with slight perivascular infiltration. From a study of this and related cases, Spiller concludes that Landry's paralysis may be due to poliomyelitis; that poliomyelitis may be due to a variety of organisms, and that inflammation occurs in the meninges and dorsal horns as well as in the ventral horns; that meningitis is not uncommon; that poliomyelitis in the adult is the same disease as in the child; that it bears a close relationship to non-purulent encephalitis and to poliencephalitis superior. With these deductions we must essentially agree.

The chief interest of this valuable study of Spiller's is the demonstration that widespread inflammation of the central nervous system may occur, leading even to a meningitis, and giving rise chiefly to motor disorders not confined to the cord. The analogy to uncomplicated poliomyelitis is apparent, and the differences are significant of the wide association poliomyelitis may have with inflammatory disorders of other portions of the nervous system.

The following case is a striking clinical example of this fact:

Case VII.—A man of twenty-seven, formerly able-bodied, after an attack of influenza, was paralyzed in both arms. This paralysis improved somewhat, leaving a high degree of muscular atrophy, without disordered sensation. Associated with this paralysis of the arms was a complete paralysis of the external rectus on the right; a partial paralysis on the left; paralysis of the right motor fifth nerve, with some disorder of sensation in the distribution of the sensory branches; paralysis of all branches of the right seventh; deafness on the right; hypoglossal and vago-glossopharyngeal group also involved to a certain degree. The patient when last seen was improving slightly.

In this case there was clearly an inferior poliencephalitis with a cervical poliomyelitis.

Sub-acute and Chronic Poliomyelitis; Progressive Muscular Atrophy:—That poliomyelitis in the adult may take several days for its complete development has already been stated; a more sudden onset than this is rare. This is, no doubt, to be explained by the greater natural resistance of the adult nervous system to toxic influences than the child's. Even a very acute meningitis or pneumonia takes a certain time for its development, and there is no reason whatever why the same should not be true in the ventral horns of the cord. The virulence of the infection must influence the rapidity of development of the symptoms. Before any adequate explanation of this may be expected we must have a far more detailed knowledge of etiology than is now at hand. In the meantime on the clinical and pathological side a few cases have been described on such good authority that the types of sub-acute and chronic poliomyelitis have been introduced into the nomenclature. In 1888, Oppenheim[65] described a case of three years' duration in a woman of about fifty, in which death finally resulted in consequence of a gradual increasing paralysis, beginning in one arm. There was loss of ventral nerve-cells, without atrophy of ventral roots or peripheral nerves. The lesions were sharply localized in the ventral horns. Before this in 1882, Eisenlohr[66] expressed himself as confident of the existence of a true sub-acute and chronic poliomyelitis. In 1893 Goldscheider[67] in an exhaustive paper discusses the subject of chronic poliomyelitis, regarding its inflammatory character as doubtful. In general it would appear hardly necessary to separate these cases of gradual onset and fatal termination from the group of progressive muscular atrophy to which they correspond much more closely than to poliomyelitis as ordinarily understood. In the absence of evidences of inflammation this attitude is still further justified, admitting always the possibility of transitional forms which must ultimately be classified on an etiological basis.

From the foregoing discussion of the pathological anatomy of poliomyelitis, it must be evident that the disease, as seen especially in the adult, represents simply a favorite location of an inflammatory process. Sufficient evidence is now at hand to prove that whether cerebral or spinal, the acute inflammations are identical in their histological manifestations, and that we are forced to the conception of poliomyelitis merely as one of a series of more or less sharply localized inflammations.

Etiology:—Speculations regarding the cause of these widespread inflammatory reactions affecting at times every part of the nervous system, are as yet somewhat premature. It is, however, becoming more and more evident that some working basis which goes beyond the findings of the microscope is advisable, provided it does not conflict with ascertained knowledge. Such an attempt to throw light on the complex relationships of the affections we have been considering, has recently been made by Strümpell and Barthelmes in their paper to which we have alluded: "Ueber Poliomyelitis acuta der Erwachsenen und über das Verhältniss der Poliomyelitis zur Polyneuritis."[68]. After speaking of the difficulty in establishing a sharp distinction between degeneration and inflammation, and discussing the general theories of the toxic causes of pathological processes, these writers offer the following etiological classification of the idiopathic atrophic paralyses:

1. The local acute infectious inflammation of a peripheral nerve—acute local neuritis.
2. The local acute infectious inflammation in the region of the ventral horns of the cord—acute local poliomyelitis of children and adults.
3. The hematogenous—toxic motor nerve-degeneration—the so-called acute polyneuritis in its different forms of origin and distribution.
4. The acute, sub-acute and chronic exogenous (toxic?) degenerations of peripheral motor neurones, including the motor spinal ganglion-cells—usually hitherto known as "poliomyelitis," sub-acute and chronic.
5. The endogenous progressive atrophy of motor neurones —neurotic and spinal progressive muscular atrophy and associated conditions (amyotrophic lateral sclerosis, etc.)

Such a classification, imperfect as it must necessarily be, has the merit of placing what exact knowledge we have on a more definite foundation, laying stress on the association of diseases, apparently dissimilar rather than in exaggerating their superficial clinical differences. The nature of the toxemia, and the reasons for its selective action remain wholly beyond the field of our present knowledge. It is a possible assumption and in a measure, at least, justified by the facts, that the striking differences between degeneration on the one hand (progressive muscular atrophy), and violent inflammations on the other (acute poliomyelitis), lie in the intensity as well as in the character of the exciting toxic cause plus the vulnerability of the affected individual. According to this grouping likewise, adult poliomyelitis simply occupies one position in a wide series of related conditions.

Conclusions:—The following conclusions as a practical clinical guide and as a working basis for further study seem justified.

That adult poliomyelitis is a well-marked clinical affection, characterized by initial fever, rapid onset of usually extensive paralyses, motor in type, with a tendency toward recovery, though often resulting fatally, from respiratory paralysis. That the disease has frequently been confused with multiple neuritis and so-called Landry's paralysis.

That its anatomical basis is a primary inflammation in the distribution of the ventral arteries of the cord, leading to a destruction of nerve-cells.

That this inflammation is rarely sharply limited to the ventral horns, but extends into the dorsal gray matter, the surrounding white matter and at times into the oblongata.

That there is no sharp line to be drawn between these lesions and still more extensive ones giving rise to totally different clinical pictures *e.g.*, encephalitis, poliencephalitis, poliencephalomyelitis.

That, therefore, anatomically the disease is much less sharply characterized than it is clinically. That its final place must be determined by a study of its cause or causes, as related to various other degenerations and inflammations of the nervous system.

That from the practical point of view it is well to consider those cases poliomyelitis which show a flaccid atrophic paralysis of sudden onset, with definite anatomical changes limited to the ventral horns of the cord and their immediate vicinity.

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SUMMARY OF CASES OF POLIOMYELITIS REPORTED IN THE LITERATURE.

In the following list of titles the important literature of poliomyelitis is given, with special reference to its occurrence in the adult, and to the pathological anatomy both in adults and children, together with certain related collateral papers. No attempt has been made to collect minor clinical reports. Various important papers bearing indirectly on the subject of poliomyelitis, especially in the adult, have recently appeared under the title of Landry's Paralysis, or Acute Ascending Paralysis. These are referred to in the foregoing text.

Duchenne: "Electrisation localizée," 1st ed., 1855.

Heine: "Beobachtungen über Lähmungszustände der unteren Extremitäten." Stuttgart, 1840.

Also: "Ueber spinale Kinderlähmung," Stuttgart, 1860.

Cornil, V.: "Paralysie infantile; cancer des seins; autopsie: altérations de la moelle épinière, des nerfs et des muscles; généralizations du cancer." Comptes Rend. de la Soc. de Biol., 187, 1863.

The first case reported with autopsy; onset in child of 2; death at 49. Loss of nerve cells and atrophy of muscles noted.

Duchenne (de Boulogne) fils: "De la paralysie atrophique graisseuse de l'enfance." Arch. gén. de Méd. II, 28, 1864. Clinical report of cases. Teeth regarded as important in etiology.

Prevost: "Observation de paralysie infantile; lésion des muscles et de la moelle." Comp. rend. de la Soc. Biol., 215, 1865.

Woman of 78: poliomyelitis in infancy. Loss of nerve cells; overgrowth of neuroglia, asymmetry of cord; reduction in size of left ventral horn.

Johnson and Clarke: "On a Remarkable Case of Extreme Muscular Atrophy with Extensive Disease of the Spinal Cord. Medico-chirurg. Trans. Lond., li, 250, 1868.

Man of 32, the disease dating from first year. Good description of pathological changes; artefacts misinterpreted. Opinion advanced that atrophy of cells is the cause of changes in the muscles.

Cuming: "Case of Extensive Paralysis from Morbid Condition of the Spinal Cord, Probably Congestion." Dublin Quart. Jour. of Med. Sciences, xlvii, 471, 1869.

Case without autopsy, in man of 40; possible alcoholic neuritis.

Charcot, Joffroy: "Cas de paralysie infantile spinale avec lésions des cornes anterieures de la substance grise de la moelle épinière." Arch. de Physiol. iii, 134, 1870.

Poliomyelitis at 7, death at 40, atrophic changes in ventral horns; degeneration of ventral nerve roots. The theory advanced that ventral nerve cells are specifically and first involved, as in progressive muscular atrophy, but more suddenly.

Parrot, Joffroy: "Note sur un cas de paralysie infantile." Arch. de Physiol. iii, 309, 1870.

Autopsy, showing loss of nerve cells and atrophy of one ventral horn with vascular infiltration.

Roger, Damaschino: "Récherches anatomo-pathologiques sur la paralysie spinale de l'enfance." Gaz. méd. de Paris, xxvi, 457, 480, 505, 541, 576; 1871.

Roger, Damaschino: Same title, Comp. rend. de la Soc. Biol. 49, 1871.

Elaborate paper, with a review of previous literature. Three cases in children reported with autopsies 2, 6 and 13 months after onset. General conclusions that lesion is primary in the cord, and secondary in muscles and nerves; that the process is an inflammatory softening of the nature of a myelitis.

Förster: Jahrbuch f. Kinderheilk. Neue Folge, V, 118, 1872.

Brief statement of two autopsies in infants; changes in peripheral nerves in one, and in cells and nerves in the other.

Chalvet: Thèse de Paris, 1872.

Gombault: "Note sur un cas de paralysie spinale de l'adulte, suivi d'autopsie." Arch. de Physiol., v, 80, 1873.

First adult case with autopsy reported. Woman of 67, typical sudden onset with fever followed by extensive paralysis of the extremities; death at end of 7 years. Post-mortem showed involvement of ventral horns, some degeneration of ventral roots, and slight involvement of oblongata; lesions not regarded as primarily inflammatory.

Petitfils: "Considérations sur l'atrophie aigue des cellules motrices (Paralysie infantile spinale; paralysie spinale aigue de l'adulte)." Paris, 1873.

Roth: "Anatomischer Befund bei spinaler Kinderlähmung." Virchow's arch., lviii, 263, 1873.

Child of 2, death 11 months after onset. Usual changes in ventral horns; dorsal horns and white matter slightly involved.

Bernhardt, M.: "Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener." Arch. f. Psych. iv, 370, 1874.

Three typical cases in adults; no autopsies. Allusion to the fact that the four extremities are usually affected in the adult.

Frey: "Ueber temporäre Lähmungen Erwachsener, die den temporären Spinalähmungen der Kinder analog sind und von Myelitis der Vorderhörner anszugehen scheinen." Berl. klin. Woch., xi, 3, 13, 28, 1874.

Clinical report of cases of temporary paralysis, with reference to observations of Kussmaul, Deutsch, Arch. f. klin. Med., i, 506, 1866.

Frey: "Ein Fall von subacute Lähmung Erwachsener—wahrscheinlich Poliomyelitis anterior subacuta." Berl. klin. Woch., xi, 549, 566, 1874.

Erb: "Ueber acute Spinallähmung (Poliomyelitis anterior acuta) bei Erwachsenen, und über verwandte spinale Erkrankungen." Arch. f. Psych. v, 758, 1874.

Valuable clinical paper with report of seven cases. Objection raised to the diagnosis "acute ascending paralysis," in many cases probably poliomyelitis.

Webber, S. G.: "A Contribution to the Study of Myelitis." Proc. Am. Neurolog. Ass'n, 55, 1875.

Alludes to involvement of white matter in poliomyelitis. Deprecates use of term, "acute ascending paralysis."

Leyden: "Beiträge zur pathologischen anatomie der atrophischen Lähmung der Kinder und der Erwachsenen." Arch. f. Psych. u. Nervenkrankh., vi, 271, 1875.

Four cases reported with late autopsies; one case without autopsy, and others quoted. Various pathological processes described, the identity of which is not definitely made out.

Cornil, Lépine: "Sur un cas de paralysie générale spinale antérieure subaigue, suivi d'autopsie." Gaz. med. de Paris, No. 11, 127, 1875.

Man of 27, gradual onset, so-called subacute poliomyelitis. Alterations found in cord.

Schultze, F.: "Zur Lehre von der spinalen Kinderlähmung und der analogen Lähmung der Erwachsenen." Virchow's Arch. lxviii, 128, 1876.

Onset at 3; death at 23. Changes in lateral tracts and Clarke's columns, in addition to usual alterations, believed to be the result of an inflammatory process. A second case, with autopsy, resembling in early course a rapid Landry's paralysis.

Dejerine: "Note sur deux cas de paralysie infantile." Le Prog. méd. vi, 423, 1878.

Two cases with autopsy, in children, believed to confirm Charcot's view of the parenchymatous nature of the disease.

Taylor, F.: "Spinal cord from a Case of Infantile Paralysis."

Trans. Patholog. Soc. Lond., xxx, 197, 1879. Description of usual changes.

Turner, F. C.: "A Portion of the Spinal Cord, with Drawings and Microscopical Specimens, from a Case of Acute Anterior Poliomyelitis in a Child, Fatal within Six weeks from the Onset." Trans. Patholog. Soc. Lond., xxx, 202, 1879.

Child of 2½; typical inflammatory changes found; some involvement of dorsal horns.

Humphrey, H.: "Case of Infantile Paralysis." Trans. Patholog. Soc. Lond., xxx, 211, 1879.

Case with autopsy in a child; duration of disease about 2 years.

Müller, F.: "Die acute atrophische Spinallähmung der Erwachsenen." Stuttgart, 1880.

Eisenlohr: "Zur Pathologie und pathologischen Anatomie der spinalen Kinderlähmung." Deutsch. Arch. f. klin. Med., xxvi, 557, 1880.

Two cases, infants; autopsies 6 and 12 months after onset. Typical lesions; slight involvement of dorsal horns and white matter.

Rank: "Zur Lehre von der Poliomyelitis anterior acuta adultorum." Deutsch. Arch. f. klin. Med., xxvii, 129, 1880.

Two cases, without autopsy; elaborate electrical examinations. Summary of knowledge up to that time.

Leyden: "Ueber Poliomyelitis and Neuritis." Ztschft. f. klin. Med. i, 400, 1880.

Important and exhaustive paper. Attempt to differentiate three forms of the disease, one of which is a peripheral neuritis.

Sange: Thèse de Paris, 1881.

Bramwell, B.: "Microscopical Preparations and Drawings Demonstrated at Meeting of Med. Chirurg. Soc. of Edin. of Pathlogy of Infantile Paralysis." Edin. Med. Jour., xxvi, 745, 1881.

Well illustrated paper, showing characteristic changes in a child's cord.

Eisenlohr: Poliomyelitis anterior subacute cervicalis circumscripta beim Erwachsenen." Neurolog. Ctbltt., 1, 409, 1882.

Man of 52; slowly progressing palsy of five years' standing. Postmortem, cord showed atrophy of ventral horn cells. E. is positive of existence of true subacute and chronic poliomyelitis.

Putnam, J. J. "Examination of the Spinal Cord in a Case of Poliomyelitis of the Adult, of Two months' Standing." JOURNAL OF NERVOUS AND MENTAL DISEASE, NO. 1, 14, 1883.

Important case; probably better classed as a "diffuse myelitis." [See text of this paper.]

Archambault, Damaschino: "Récherches cliniques et anatomo-pathologiques sur un cas de paralysie spinale de l'enfance, avec autopsie au vingt-sixième jour de la maladie." Rev. mensuelle des mal. de l'enfance. I, 63, 1883.

"Red softening" in ventral horns.

Sahli, H.: "Zur Lehre von der spinalen Localization. Sectionsbefund bei einer alten Kinderlähmung mit eng localizirter Atrophie." Deutsch. Arch. f. klin. Med., xxxiii, 360, 1883.

Schultze: "Ueber aufsteigende atrophische Paralyse mit tödtichem ausgange." Berl. klin. Woch., xx, 593, 1883.

A case of subabcute onset in a syphilitic of 44; death in 6 months; no autopsy. A second case of insidious onset in a man of 30, death with increasing motor weakness in 8 months. No autopsy. Interesting discussion of Landry's Paralysis.

Money: "The Spinal Cord of a Recent and of an Old Case of Infantile Palsy." Trans. Patholog. Soc. Lond., xxxv., 45, 1884.

Two cases, with unusual changes post-mortem. Fanciful theory offered in explanation of the alterations.

Drummond, D.: "On the Nature of the Spinal Lesion in Poliomyelitis Anterior Acuta, or Infantile Paralysis." Brain, viii, 14, 1885.

Child of 5; death 6 or 7 hours after onset. Vascularity of ventral horns, but not sharply confined to them; minute hemorrhages; free leucocytes; nerve cells swollen, granular and ill-defined.

Immermann: "Ueber Poliomyelitis acuta und Landry'sche Paralyse." Neurolog. Ctbltt. iv, 304, 1885.

Man of 22: ascending motor paralysis of the clinical type of Landry's Paralysis. Death; inflammation of ventral horns only.

Friedländer, C.: "Ueber Verkalkung der Ganglienzellen." Virchow's Arch., lxxxviii, 84, 1888.

Three cases with autopsy, one in an adult. Finds a process of calcification of nerve cells which is regarded as a constant change in acute poliomyelitis. See also Virchow's Arch., xi., 620 and l, 304.

Rissler, J. "Zur Kenntniss der Veränderungen des Nervensystems bei Poliomyelitis anterior acuta." Nordiskt med. arkiv., xx, No. 22, 1, 1888.

Five cases, with autopsies, 3 early, 2 old, 1 an adult with death on the eighth day. Conclusion that in acute cases there is cellular exudation and degeneration of nerve cells, but, agreeing with Charcot, that the cellular changes are primary. A long, unnecessarily tedious, but very important paper.

Oppenheim: "Ueber die Poliomyelitis anterior chronica." Arch. f. Psych., xix, 318, 1888.

Gradual motor paralysis in a woman of 53, increasing over a period of three years. Loss of ventral nerve cells, without corresponding atrophy of ventral nerve roots and peripheral nerves. Important case in connection with so-called chronic poliomyelitis.

Kawka: Halle Dissertation, 1889.

Joffroy, Achard: "Contribution a l'anatomie pathologique de la paralysie spinale aigue de l'enfance." Arch. de méd. expériment., i, 57, 1889.

Two cases of poliomyelitis in infancy; death in old age. Typical lesions, including changes in peripheral nerves, muscles and bones.

Rosenberg: Heidelberg Dissertation, 1890.

Williamson: "The Early Changes in the Spinal Cord in Acute Anterior Poliomyelitis of the Adult." Med. Chron., Manchester, xii, 454, 1890.

Man of 22; death at end of five weeks. Inflammatory changes in ventral horns. No microorganisms found.

Medin: Ueber eine Epidemic von spinaler Kinderlähmung." Verhandl., x, Med. Cong. Berlin, II, Abt. vi, 37, 1891.

Epidemic in Stockholm.

Also: Hygiea, xlii, 657, 1890, and abs. in Neurolog. Ctbltt., 397, 1891.

Oppenheim: "Zur Pathologie der chronichen atrophischen Spinallähmung." Arch. f. Psych., xxiv, 758, 1892.

Von Kahlden: "Ueber Entzündung und atrophie der Vorderhörner des Rückenmarks." Ziegler's Beiträge, xiii, 113, 1893.

Reversion to Charcot's view of primary change in nerve cells.

Goldscheider: "Ueber Poliomyelitis anterior." Deutsch. med. Woch., xix, 441, 1893.

Goldscheider: "Ueber Poliomyelitis." Ztschrift. f. klin. Med., xxiii, 494, 1893.

Covers ground of former paper more completely. Opposes Charcot's view but regards inflammatory character of chronic poliomyelitis as doubtful. Best review of the whole subject and allied conditions up to this date.

Lippmann: "Poliomyelitis anterior." Deutsch. med. Woch., xix., 823, 1893. Discussion of Goldscheider's first paper, with report of a case in a child, with autopsy.

Dauber: "Zur Lehre von der Poliomyelitis anterior acuta." Deutsch. Ztschft. f. Nervenheilk. iv, 200, 1893.

Case of a child; autopsy, with inflammatory changes in cord.

Redlich: "Beitrag zur pathologischen anatomie der Poliomyelitis anterior acuta infantum." Wien. klin. Woch., vii, 287, 1894.

Infant; death in ten days; extensive inflammatory changes. Suggests that poliomyelitis and polyneuritis are same affection, with different localizations.

Siemerling: "Zur pathologischen Anatomie der spinalen Kinderlämung." Arch. f. Psych., xxvi, 267, 1894.

Two acute cases in infants; usual inflammatory changes found, with secondary nerve cell alterations.

Caverly: "History of an Epidemic of Acute Nervous Disease of Unusual Type." N. Y. Med. Rec., xlvi, 673, 1894.

A revision of this paper was later published in pamphlet form with the title: "History of an Epidemic of Acute Anterior Poliomyelitis."

In this epidemic the farm animals were also attacked. There were about 125 cases, chiefly in children; the brain was frequently involved. There were no autopsies.

Mund: Erlangen Dissertation, 1897. Same case as reported by Strümpell in 1900.

Grawitz: "Ein Fall von Poliomyelitis anterior subacuta mit Zwerckfelllähmung bei einer Erwachsenen." Berl. klin. Woch., xxxiii, 245, 1896.

Woman of 18, involvement of all extremities, of rather gradual onset. The patient did not die.

Bicknel: "Ein Fall von acuter Poliomyelitis beim Erwachsenen unter dem Bilde der aufsteigenden Paralyse."

Bonn: Dissertation, 1898.

Edward: Thèse de Paris, 1898.

Niedner: "Ein Fall von Poliomyelitis Acuta der Erwachsenen." Münch. med. Woch., xl, 566, 1898.

Previously healthy man of 19; sudden onset; death in three days; no autopsy.

Matthes: "Sectionsbefund bei einer frischen spinalen Kinderlähmung." Deutsch. Zeitschrift f. Nervenheilk, xiii, 331, 1898.

Infant; death in eight days. First description of changes by use of Nissl Method.

Schultze: "Zur Aetiologie der Acuten Poliomyelitis." Münch. Med. Woch. xlv, 1197, 1898.

Child of five;; possible coincident meningitis and poliomyelitis. Weichselbaum diplococcus found in cerebro-spinal fluid; no autopsy.

Jagiç: "Zur Kenntnis der acuten Poliomyelitis der Erwachsenen" (Acute aufsteigende Spinallähmung). Wien. med. Woch., xlix., 394, 1899.

Man of 28, simulation of Landry's Paralysis; inflammatory changes of cord and oblongata.

Bülow-Hansen, Harbitz: "Beitrag zur Lehre der acuten Poliomyelitis." Ziegler's Beiträge, xxv, 517, 1899.

Three children in same family, two deaths with autopsy and usual findings.

Dercum: "Note on a Case of Acute Poliomyelitis in Which the Cerebro-spinal Fluid Obtained by a Quincke Puncture contained a diplococcus, resembling the diplococcus of Sternberg." JOURNAL OF NERVOUS AND MENTAL DIS., xxvii, 116, 1900.

Hoppe: "Poliomyelitis anterior acuta adultorum." Jour. Am. Med. Ass'n, xxxiv., 80, 1900.

Report of two cases observed clinically.

Sherman and Spiller: "A Case of Poliencephalomyelitis in an Adult. Presenting the clinical picture of Landry's Paralysis and fatal in 38 hours after first definite motor symptoms." Phil. Med. Jour., v, 734, 1900.

Man of 21, rapid ascending motor paralysis; death from paralysis of respiration; widespread changes of inflammatory character throughout the central nervous system. One of the most important published cases.

Strümpell, Barthelmes: "Ueber Poliomyelitis acuta der Erwachsenen and über das Verhältniss der Poliomyelitis zur Polyneuritis." Deutsch. Ztschft. f. Nervenheilk., xviii, 304, 1900.

An important paper; for details see text.